EDITORIALS

Hypothermia

HYPOTHERMIA IS DEFINED as a clinical condition in which the deep body temperature falls below 35°C (95°F). As recently as 20 years ago it was thought to be a rare condition, but since the early 1960's it has become recognized as a problem particularly affecting elderly people. Hospital records have indicated that very few cases are recognized clinically before admission, but a survey carried out by the Royal College of Physicians of London[1] in ten hospitals in England and Scotland during three months in the winter of 1965 showed that 0.68 percent of all patients admitted had hypothermia. Of these, 42 percent were older than 65, and the incidence in this age group was 1.2 percent. Ten years later a further study by Goldman and her colleagues[2] showed that hypothermia occurred in 3.6 percent of all patients older than 65 who were admitted to hospital.

Many factors are responsible for the greater liability of old people to the development of hypothermia. In a population study conducted by Fox and his colleagues,[3] 10 percent of elderly people living at home were found to be on the border line of hypothermia, with a deep body temperature (as measured by urine temperature) of less than 35.5°C (95.9°F). These persons were thought to have some degree of thermoregulatory failure as shown by the inability to maintain an adequate core-peripheral temperature gradient. Further investigation of this impairment of thermoregulatory control by Collins and co-workers[4] showed that abnormal patterns of peripheral blood flow on cooling accompanied by a failure of vasoconstriction were much more common in old people than in young. When tests of thermoregulatory function were repeated four years later with the same subjects[4] a significantly higher proportion had low peripheral blood flow and a higher proportion had a nonconstrictor response on cooling. Postural hypotension with a fall in systolic pressure of 20 mm of mercury or more on standing occurred in 14 percent of old people—again an indication of impairment of autonomic function in old age. In addition, many old people have a diminished sensitivity to cold. Tests of digital thermosensation show that young people can perceive mean temperature differences of about 0.8°C whereas older persons can discriminate only between mean temperature differences of 2.1°C and some are unable to perceive temperature differences of 5.0°C or more.[4] It is likely that a lessened sensitivity to cold is one of the reasons for the relatively large number of old people who appear to be able to tolerate cold conditions without discomfort. Nevertheless, such persons may be at risk of overtaxing the heat-conserving capacity of a failing thermoregulatory system.

The common clinical conditions associated with hypothermia in the elderly are bronchopneumonia and other severe infections, cardiac infarction and pulmonary embolism, which can cause an acute derangement of thermoregulatory mechanisms. Myxedema and diabetes mellitus are the most frequent endocrine disorders causing hypothermia. In persons with diabetes, hypothermia can complicate hyperglycemic ketoacidotic coma and hyperosmolar nonketotic coma; but in elderly persons, an important factor is the presence of autonomic dysfunction associated with diabetic peripheral neuropathy. In several neurological and locomotor disorders immobility can limit the amount of heat generated, and in parkinsonism there may be an additional factor of disturbance of autonomic function. Patients with confusional states and dementia may be unaware of environmental hazards and may not adequately protect

themselves against the cold. The psychotropic drugs, especially phenothiazine tranquillizers, prescribed for these conditions may also affect thermoregulation. Some of these drugs lead to hypothermia by the abolition of shivering and by impairing the normal centrally mediated vasoconstrictor response to cold.

Acute alcohol intoxication and chronic alcoholism are well-recognized causes of hypothermia. In the series reported by Goldman and colleagues[2] alcohol was implicated in 6 of 39 elderly patients with low body temperatures admitted to hospital. These patients are often vagrants who have been subjected to cold out-of-doors. Alcohol causes hypothermia mainly by inducing vasodilatation, but it also reduces shivering and depresses central thermoregulatory control. In addition, alcohol can induce hypoglycemia, especially when taken after exercise and on an empty stomach. Haight and Keatinge[5] found that the ingestion of 28 ml of alcohol by fit young men lowered the blood glucose level to 40 mg per dl. Following exposure to cold air for half an hour the blood glucose level fell even further, and deep body temperatures dropped by 2.8°C (5°F). When alcohol ingestion was not a factor, fasting, exercise and exposure to cold affected neither the body temperature nor the blood glucose level.

In the August issue Dr. Faith Fitzgerald of the University of Michigan Medical School reported findings in 22 hypothermic patients who, with the exception of 2, were alcoholics [Fitzgerald FT: Hypoglycemia and accidental hypothermia in an alcoholic population. West J Med 133:105-107, Aug 1980]. Nine patients (41 percent) had serum glucose levels of less than 50 mg per dl. Fitzgerald emphasizes the importance of blood glucose estimation and administration of glucose intravenously in these alcoholic hypothermic patients who are found to be hypoglycemic. Philip and Smith[6] have drawn attention to the often overlooked association between hypothermia and Wernicke encephalopathy. The petechial hemorrhages that occur in the walls of the third ventricle, the hypothalamus and mammillary bodies are probably responsible for the impaired temperature regulation in this condition, which in turn is caused by a thiamine deficiency, often in association with alcoholism. If recognized early enough the response to intravenous administration of thiamine is usually dramatic.

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ACCME

IT APPEARS that the rift between the American Medical Association and the Liaison Committee on Continuing Medical Education (LCCME) has ended, and that there will now be a single body to be called the Accreditation Council for Continuing Medical Education (ACCME). This is a welcome turn of events and much credit goes to the principals concerned who simply went ahead and did what needed to be done. The new body will be relatively unfettered and it has a clear mandate from its seven sponsoring organizations to do what needs to be done for the accreditation of continuing medical education (CME).

CME is emerging as a very major segment of medical education and its dimensions are quite different from undergraduate education or residency training. Undergraduate medical education seeks to give a student a broad framework upon which to hang a growing knowledge of medicine. It generally occurs in just one kind of setting, a medical school. Residency training enables a postdoctoral student to master a specialty which may be as broad as family practice or as narrow as nuclear medicine. It may occur in one or several settings. Although it has roots in broad general medicine and in a physician's specialty, effective CME relates much more closely to the further differentiated practice of an individual physician, and is at its best when it addresses the kind of problems that a physician sees or may see in that practice. And then CME takes place in many, many kinds of settings.

Accreditation of CME is an important and complex task. There must be minimum standards and there must be accountability. Also there must be enough flexibility to take into consideration many kinds of learning in many different situations. And